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Chapter 7: Cardiac Arrest

INTRODUCTION

- *Cardiac arrest* is the cessation of cardiac mechanical activity, confirmed by absence of circulation signs (eg, detectable pulse, unresponsiveness, apnea).

CLINICAL PRESENTATION

- Cardiac arrest may be preceded by anxiety, crushing chest pain, nausea, vomiting, and diaphoresis.
- Signs include apnea; hypotension; no detectable pulse; cyanosis; cold, clammy extremities; loss of consciousness; and syncope.

DIAGNOSIS

- Rapid diagnosis is crucial and based on observing clinical manifestations consistent with cardiac arrest.
- Electrocardiography (ECG) identifies the cardiac rhythm, determining drug therapy.

  ✓ Ventricular fibrillation (VF) is electrical anarchy of the ventricle resulting in no cardiac output and cardiovascular collapse.

  ✓ Pulseless electrical activity (PEA) is absence of a detectable pulse and presence of electrical activity other than VF or pulseless ventricular tachycardia (PVT).

  ✓ Asystole is no electrical heart activity, indicated by a flat line on the ECG.

TREATMENT

- Goals of Treatment: Resuscitation goals include preserving life; restoring health; relieving suffering; limiting disability; and respecting the individual's decisions, rights, and privacy. This is achieved through cardiopulmonary resuscitation (CPR) and return of spontaneous circulation (ROSC) with effective ventilation and perfusion to minimize hypoxic organ damage. The primary outcome after resuscitation is survival to hospital discharge with good neurologic function.

GENERAL APPROACH

- The online American Heart Association (AHA) CPR and emergency cardiovascular care (ECC) guidelines are updated continuously and emphasize timely implementation of the "chain of survival."

  ✓ Out-of-hospital arrests: (1) early recognition of cardiac arrest and activation of the emergency response system, (2) high-quality CPR, (3) rapid defibrillation, (4) advanced cardiac life support (ACLS), (5) post-arrest care, and (6) recovery.

  ✓ In-hospital arrests: (1) early recognition and prevention of cardiac arrest, (2) activation of emergency response, (3) high-quality CPR, (4) prompt defibrillation, (5) post-arrest care, and (6) recovery.

Basic Life Support Given by Healthcare Providers

- The CPR sequence follows the CAB mnemonic: circulation, airway, breathing. Basic life support by *trained healthcare providers* includes:

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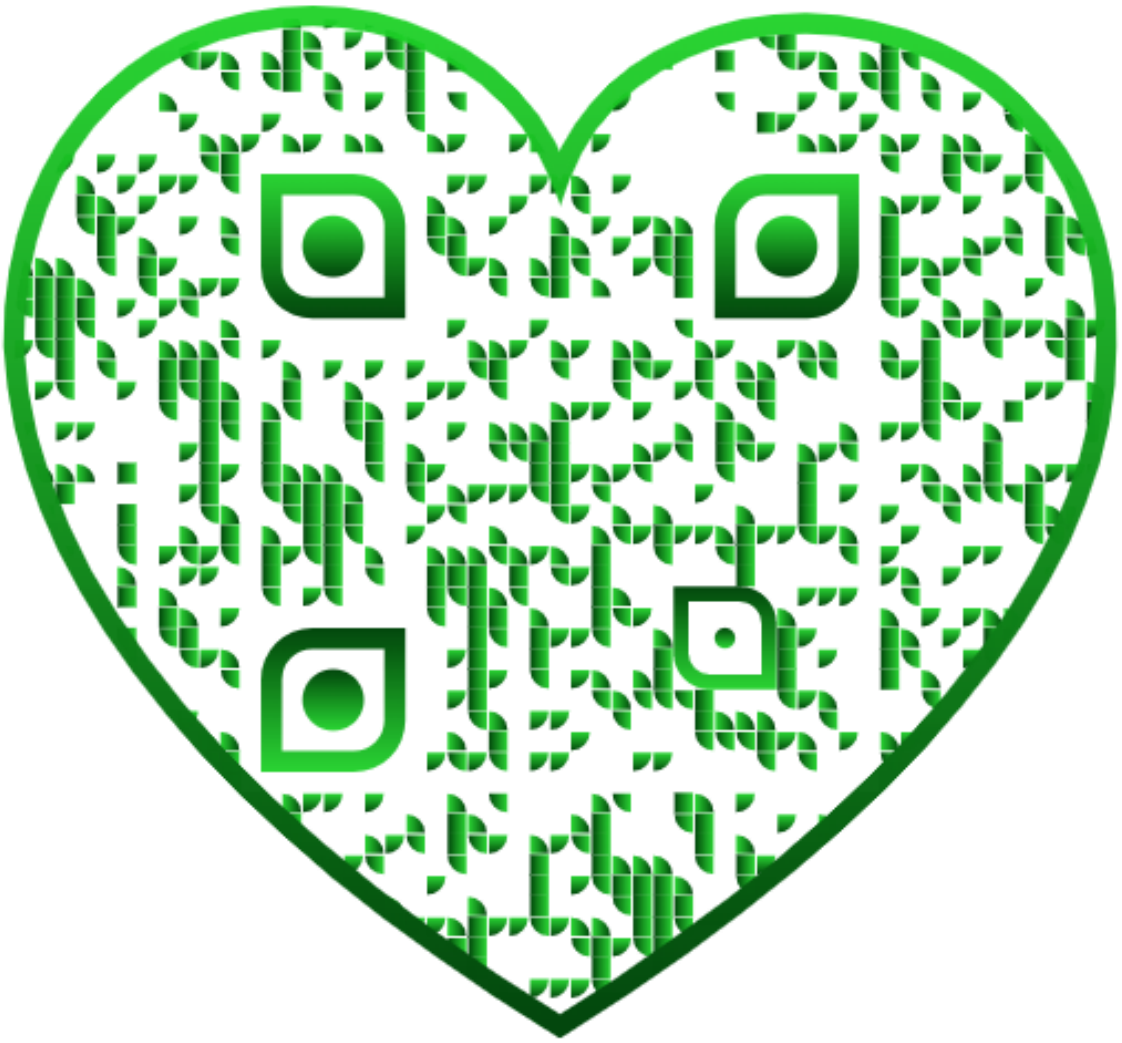

✓ Determine patient responsiveness. If unresponsive with no breathing or no normal breathing (ie, only gasping), activate the emergency medical response team and obtain an automated external defibrillator (AED).

✓ Check for a pulse. If not definitely felt within 10 seconds, begin CPR with chest compressions and use AED when available.

✓ Begin CPR with 30 chest compressions at a rate of 100–120/min, compressing at least 2 in (50 mm) deep in adults.

✓ Open the airway and deliver 2 rescue breaths, then repeat chest compressions. Follow each 30 chest compressions with 2 breaths.

✓ Continue CPR until an AED is ready for use or ACLS providers take over care.

✓ When an AED is available, check rhythm to determine if defibrillation is advised. If so, deliver one shock then resume chest compressions/rescue breaths for 2 minutes or until the AED prompts a rhythm check.

✓ If the rhythm isn't shockable, immediately resume CPR for 2 minutes or until prompted by AED. Continue until ACLS providers take over or victim starts to move.

Advanced Cardiac Life Support

- Once ACLS providers arrive, definitive therapy follows the ACLS algorithm shown in **Fig. 7-1**.
- A bag-mask device or an advanced airway (eg, endotracheal tube, supraglottic device) may be used for ventilation. One provider delivers 1 breath every 6 seconds while another performs continuous chest compressions.
- Central venous catheter administration allows for faster, higher peak drug concentrations than peripheral venous administration, but is rarely feasible during resuscitation. If a central line is already present, use is recommended. If no IV access exists, insert a large peripheral venous catheter. If unsuccessful, insert an intraosseous (IO) device.
- Endotracheal drug administration is possible, but has fallen out of favor due to unpredictable therapeutic drug levels and increased IO access availability.

FIGURE 7-1

Treatment algorithm for adult cardiac arrest: ACLS.

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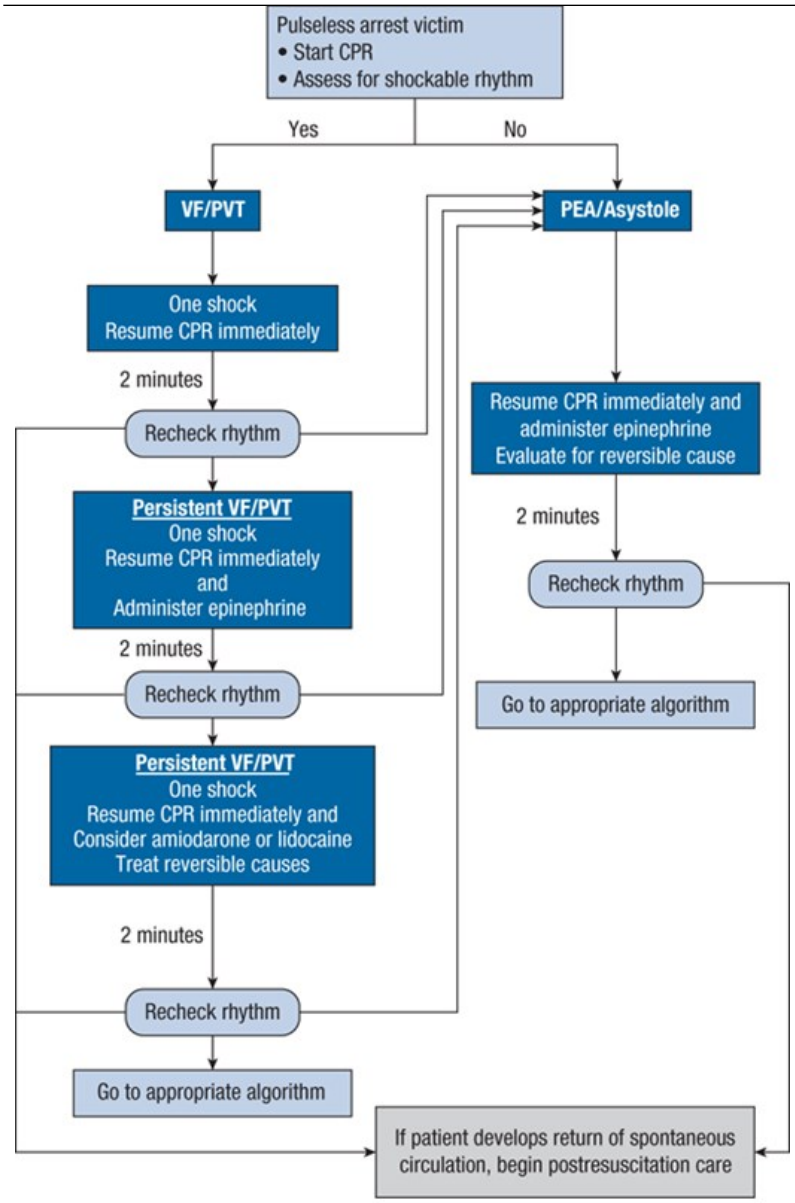

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TREATMENT OF VENTRICULAR FIBRILLATION AND PULSELESS VENTRICULAR TACHYCARDIA

Nonpharmacologic Therapy

- Defibrillation and CPR are essential for treating cardiac arrest with VF and PVT. CPR alone is unlikely to terminate VF/PVT, and defibrillation is often necessary to restore cardiac rhythm.

Pharmacologic Therapy

Epinephrine

- **Epinephrine** is the first-choice drug for treating VF and PVT. It acts as an α- and β-receptor agonist, to cause vasoconstriction and increases rate and forcefulness of heart contractions, augmenting low coronary and cerebral perfusion pressures.

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- The recommended adult dose is 1 mg IV or IO every 3–5 minutes. Higher doses have been studied, but are not routinely recommended in cardiac arrest.

Vasopressin

- **Vasopressin** is a potent nonadrenergic vasoconstrictor that increases blood pressure (BP) and systemic vascular resistance. Despite theoretical advantages, clinical outcomes are not superior to standard-dose epinephrine alone or in combination with vasopressin or methylprednisolone.

Antiarrhythmics

- Antiarrhythmic drugs after unsuccessful defibrillation and vasopressor administration prevent development or recurrence of VF and PVT. However, evidence of improved survival to hospital discharge is lacking.
- **Amiodarone** or **lidocaine** may be considered in adults with VF/PVT refractory to defibrillation and epinephrine.
- Amiodarone is administered as 300 mg IV/IO followed by a second dose of 150 mg.
- An initial lidocaine dose of 1–1.5 mg/kg IV is administered. Additional doses of 0.5–0.75 mg/kg every 5- to 10-minute intervals, up to a maximum total dose of 3 mg/kg are used if VF/PVT persists.

Magnesium

- While severe hypomagnesemia is associated with VF/PVT, routine magnesium use during cardiac arrest doesn't improve outcomes. It should only be administered for torsades de pointes. The dose of 1–2 g diluted in 10 mL of 5% dextrose in water is administered IV/IO push over 1–2 minutes.

TREATMENT OF PULSELESS ELECTRICAL ACTIVITY AND ASYSTOLE

Nonpharmacologic Therapy

- Successful treatment of PEA and asystole depends on the diagnosis of the underlying cause. Potentially reversible causes include (1) hypovolemia, (2) hypoxia, (3) acidosis, (4) hyper- or hypokalemia, (5) hypothermia, (6) hypoglycemia, (7) drug overdose, (8) cardiac tamponade, (9) tension pneumothorax, (10) coronary thrombosis, (11) pulmonary thrombosis, and (12) trauma.
- PEA and asystole are treated the same: CPR, airway control, and IV access. Avoid defibrillation because the resulting parasympathetic discharge can lower ROSC and survival chances.

Pharmacologic Therapy

- **Epinephrine** (1 mg by IV or IO injection every 3–5 minutes) is the primary pharmacologic agent for asystole and PEA and should be administered as soon as possible.

ACID–BASE MANAGEMENT

- Acidosis occurs during cardiac arrest because of decreased blood flow or inadequate ventilation. Chest compressions generate only 25% of normal cardiac output, causing inadequate organ perfusion, tissue hypoxia, and metabolic acidosis. Lack of ventilation causes CO_2 retention, causing respiratory acidosis. Acidosis reduces myocardial contractility, decreases vasomotor tone, and lowers the fibrillation threshold.
- Routine sodium bicarbonate use is not recommended because it doesn't improve ROSC or survival to hospital discharge, and may have detrimental effects. It can be used in special circumstances (eg, hyperkalemia, tricyclic antidepressant overdose, salicylate toxicity).

POSTRESUSCITATIVE CARE

- ROSC after cardiac arrest may lead to post-cardiac arrest syndrome characterized by hypoxic brain injury, myocardial dysfunction, systemic ischemia-reperfusion response, and the underlying persistent precipitating pathology.

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- Ensure adequate airway and oxygenation. After resuscitation with 100% oxygen, titrate the oxygen fraction down as tolerated to maintain an oxyhemoglobin saturation of 92%–98% (0.92–0.98).
- Immediately evaluate for ECG changes consistent with acute MI and perform revascularization if present.
- Cerebral hypoperfusion may last several hours after resuscitation; augmenting BP to achieve a mean arterial pressure (MAP) goal >80 mm Hg is recommended.
- Temperature control, previously known as therapeutic hypothermia or targeted temperature management, helps protect against cerebral injury by suppressing chemical reactions that occur after blood flow restoration. Although randomized controlled trials produced mixed results on neurologic function and survival, guidelines recommend maintaining temperature between 32°C and 37.5°C for at least 24 hours. Prevent fever afterward. Potential complications of temperature control include coagulopathy, dysrhythmias, bradycardia, diuresis, electrolyte disorders, hyperglycemia, and infection risks.

EVALUATION OF THERAPEUTIC OUTCOMES

- The optimal outcome following CPR is an awake, responsive, spontaneously breathing patient. Resuscitation is successful if the patient remains neurologically intact with minimal morbidity.
- ECG rhythm assessment and pulse checks are the only physiologic parameters to guide therapy. However, palpating a pulse during CPR has not been shown to effectively assess blood flow.
- Invasive hemodynamic monitoring (eg, coronary perfusion pressure, central venous oxygenation) can be useful but is seldom available. Arterial diastolic pressure can serve as a surrogate for coronary perfusion pressure with a suggested goal of >25 mm Hg.
- End-tidal carbon dioxide ($ETCO_2$) monitoring is a useful method to assess cardiac output during CPR and is linked to ROSC. Persistently low $ETCO_2$ values (<10 mm Hg [1.3 kPa]) during CPR in intubated patients suggest that ROSC is unlikely.
- In the postresuscitative phase, monitoring should focus on the post-cardiac arrest syndrome, as it can affect most organ systems. A full system review is recommended.
- Many cardiac arrest survivors experience prolonged emotional, cognitive, physical, and neurologic symptoms. In recovery, provide a multimodal discharge plan with instructions for treatment, rehabilitation, and surveillance; and set clear short- and long-term expectations clearly with appropriate action plans for each recovery phase.

See Chapter e44, Cardiac Arrest, authored by Jeffrey F. Barletta and Brittany D. Bissell Turpin, for a more detailed discussion of this topic.

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